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Epilepsy or stereotypy? Diagnostic issues in learning disabilities

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Approximately 0.8% of people in the general population have epilepsy. Within this group are specific sub-populations who co-present with other additional conditions, learning disability being one such example. Epilepsy rates are the highest of all in this subgroup, between 21% and 50% and positively correlated with degree of learning disability. In addition, in the more severe categories, problems frequently arise when attempting to differentiate epileptic events from other phenomenon, such as stereotyped behaviours and involuntary movements. The individual is unable to communicate changes in consciousness and perception and observers often find it difficult to detect such changes, particularly with regard to the partial epilepsies. Intensive monitoring using EEG and video equipment can often prove valuable in such a situation in assisting carers to recognize epileptic episodes and respond accordingly.

Key words: stereotypy; EEG; nonepileptic; diagnosis.

SEIZURE IDENTIFICATION

The identification and diagnosis of seizure types can be time-consuming and is rarely straightforward. There can be subtle differences between one motor manifestation of epilepsy and another. An example of this is primary vs. secondary generalized seizures. In the former an individual may quite suddenly enter the tonic and clonic phases whereas in the latter they may report a brief prior jerking of a limb without loss of consciousness, followed by the generalized phase.

Diagnosis can be greatly assisted by a detailed history of events. Indeed, Aicardi[1] stated 'It is true... that the diagnosis is as good as the history'. Prescribing decisions in routine practice rely upon detailed histories unless actual events are witnessed by the doctor (a rare phenomenon with classic epileptic seizures) or are recorded during EEG investigations.

Furthermore, such detailed descriptions can often assist in distinguishing between isolated episodes which may not be epileptic in origin, such as ischaemic attacks and non-epileptic seizures, and genuine seizure incidents[2,4].

Of course, practitioners may receive extremely detailed self-report information resembling seizure activity which is unreliable. A good example of this is Münchausen's Syndrome by proxy, where a mother may deliberately suffocate her child to induce seizure-like symptoms in order to gain attention.

If a person is able to report occurences which are not evident to the observer such as a strange smell or headache, then this can often prove to be valuable information. Where there exist additional difficulties in making a diagnosis is in cases where there are no witnesses and the patient has little recollection, or where the patients' understanding and communication abilities are limited, such as in people with a learning disability.

If the individual has a physical disability then the origins of particular behaviours which resemble epileptic phenomenon may be questioned, i.e. involuntary movements/tics, but again they may be unable to communicate enough to assist in diagnosis. Similarly for others who have specific movement disorders, communication may be limited but behaviours indicate possible seizure origin, e.g. Tourette's Syndrome.

Usually in such complex cases assistance with diagnosis can be provided by EEGs, both routine and ambulatory, as well as MRIs and CAT scans if deemed appropriate. Evidence of such investigations will not be explored at this point but

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instead will be presented later in relation to learning disabilities.

In summary, the indication is that diagnosis of a condition such as epilepsy is fraught with difficulties as external phenomena associated with a variety of origins can often be mistaken for epileptic events. There are various conditions which confuse and sometimes completely mislead the practitioner and a number of these will be explored in some detail, however the emphasis will be upon conditions associated with people with a learning disability as this group often pose a particular challenge.

DIFFERENTIAL DIAGNOSIS

Non-epileptic seizures

Nonepileptic siezures (NES) is a term assigned to behaviours which may also be referred to as pseudoseizures or psychogenic (referring to an event with an emotional/psychological origin). What the terminology actually refers to is defined by Vosslers as: '... paroxysmal events that alter or appear to alter neurologic function to produce motor or sensory, autonomic or psychic symptoms that at least superficially resemble those occuring during epileptic seizures'.

A number of investigations have attempted to distinguish between epileptic and non-epileptic events[4,6–9]. These studies range from investigations of individuals who have been sexually abused in childhood[2,3] and have apparent non-epileptic attack disorders through to specific disorders of sleep such as Periodic Limb Movement Disorders[10]. Stephenson[4] explores the confusion with a variety of other physiological events such as anoxic syncope caused by either a reduction in cerebral blood flow or oxygen content or both.

Vossler[5] also reviews a number of conditions which are also physiological events but not epileptic in origin. These include migraines which produce symptomatology similar to simple partial seizures (disorientation, amnesia) and non-REM parasomnias in children where the children are disorientated, have no recollection and perform automatisms similar to complex partial seizures.

It is of course difficult to estimate the prevalence of non-epileptic cases, however Gates[11] refers to one particular epilepsy centre where 20–30% of new referrals had non-epileptic seizures and Vossler[5] reported an NES frequency rate of 32% in 231 patients attending a Swedish medical centre. Diagnosis is often compounded by the fact that epileptic and non-epileptic events, either physiological or psychological, can and frequently do co-exist, a fact which is extremely important in implementing treatment programmes.

To highlight further confusion, Greig and Betts[8] provide detailed histories of six patients who had epilepsy but had also been sexually abused. They emphasised that new cases requiring diagnosis should not always be immediately assumed to be non-epileptic in origin if there is a history of sexual abuse. Again, the two may co-exist with no conclusive evidence of a causal relationship.

In the learning disabled population identification of non-epileptic events follows a similar procedure to that in the general population—particular attention is paid to gathering as detailed a history as possible and, where deemed necessary, an EEG may be requested[12].

Generally stereotypies and movement disorders are included in the category of non-epileptic events but they have been extracted for individual consideration as they are extremely important in this group.

Movement disorders

As previously highlighted, further confusion with epilepsy has arisen with a wide range of movement disorders including Tardive Dyskinesia. This condition is particularly prevalent in people with a mental illness where it is believed to be (in some cases) drug-induced, the result of long-term exposure to phenothiazines or butyrophenones for therapeutic purposes. Patients exhibit a variety of involuntary movements particularly in the orofacial area, which are frequently irreversible following drug withdrawal[3]. Generally these categories of drugs are also frequently prescribed for people with learning disabilities who have behaviour problems.

Huntington's Chorea is a disorder where individuals display involuntary movements sometimes deemed as stereotypic which are treated by introducing dopaminergic receptor blockers. This supports the theory that increased dopamine release produces repetitive movements. Such stereotyped behaviours have been elicited in rats

by the administration of amphetamines which release endogenous dopamine[14–16].

People with Gilles de la Tourette Syndrome display symptomatology which includes involuntary motor and vocal tics. Rickards[7] believes that confusion arises in two particular areas, (1) myoclonic jerks may mimic similar motor (and occasional) vocal tics and (2) more complex movements or vocalizations may be interpreted as partial seizures. EEG investigations were conducted on people with Tourette's Syndrome and although there was evidence of some non-specific abnormalities[18] the generalized 'paroxysmal patterns and occasional generalised background slowing of myoclonic epilepsy were absent[9].

Aside from medical investigation, behaviours exhibited by some of these distinct populations are readily differentiated by means of close external observation and (when possible) verbal communication with the patient. Unfortunately the opportunities for a reciprocal communicative relationship between physician and patient in learning disabilities are somewhat rare. Indeed a high proportion of what is reported is that which is observed by a variety of carers in a number of settings; this is obviously of prime importance but does not allow for the level of detail which can be provided by self-report.

Stereotyped behaviours

It is widely recognized that a considerable number of people with severe or profound learning disabilities display behaviours which are deemed to be stereotypic in nature. These range in form from body rocking and pacing (increased intensity and duration of so-called normal behaviours) to more complex fine motor movements, posturing and vocalizations. These behaviours are idiosyncratic, repetitive, rhythmical and intensive movements which occur frequently and are extremely persistent. They appear to be of considerable importance to the individual, often to the exclusion of all else; indeed investigations have highlighted cases where individuals will risk pain or accept loss of food in order to continue with this means of 'self-stimulation'[20].

There is evidence that these behaviours are exhibited by a considerable number of learning disabled people, as Repp[21,22] observed a frequency rate of between 7 and 47% in those residing in institutions while Walsh[23] found the rate to be around 57% in his investigations on a similar population. The most conservative estimate could be that of Dura[24] of 34% in a study population of 102 non-ambulatory severely learning disabled people, however study populations vary to the extent that it is extremely difficult to generalize.

Often stereotyped behaviours are characteristic and enduring features of the individual's presentation and depending upon the care circumstances and care aims may or may not present a challenge to management. However with recent changes in legislation and transfer of people from long-stay institutions to community housing, it appears that these performances and their typology can have considerable implications for the overall care of an individual. In some cases it has been deemed necessary to implement specific aversive therapies as a form of treatment to abolish such displays. These treatments have included physical restraint and sprays of fine water mist.

The rationalization behind such treatments is often two-fold, one, that such behaviours prevent an individual from being accepted in the community at large and thus treatments are necessary to improve quality of life. Two, intervention is often implemented because of a concern that such behaviours may develop into self-injurious displays (SIB). In some cases neuroleptic medication such as thioridazine has been prescribed to reduce these behaviours. Yet considerable criticism could be made of the fact that a number of these treatments have been implemented without any real understanding of the function of such behaviours. Such movements could actually be the external manifestation of neurological damage, damage which is recognizable by the incessant nature of the movements or indeed by their peculiarity.

For example, some of the behaviours witnessed may be regarded as an exaggeration of normal behaviours (such as rocking or pacing the floor), but generally a high proportion of that which is observed is not instantly recognizable as an exaggerated normal behaviour, i.e. flicking fingers directly in front of face whilst making a clucking sound or observing the world through the formation of the letter 'c' using the thumb and forefinger. In these cases, where behaviours may be the direct result of neurological damage, it is unclear as to whether any particular interventions would be of any benefit. For example, can specific techniques to modify behaviour compensate for brain damage? Can negatively reinforcing such

displays really prevent or decrease them in the long term?

EPILEPSY AND STEREOTYPY IN LEARNING DISABILITIES

Epilepsy, as previously mentioned, is a condition which is present in a high proportion of learning disabled people. For those who are deemed to be mildly or moderately learning disabled epilepsy frequency rates are estimated at 21%[25]. Bicknell[26] suggests rates of between 40 and 50% in others with profound and multiple learning disabilities with the epilepsy being positively correlated with severity of disability. With regard to antiepileptic drug treatment, 53% of children and adults with a learning disability are in receipt of at least one such agent[24] polypharmacy being implemented in 40% of cases[27,28].

Frequently, language has been used to describe epilepsy which embraces similar terminology to that which is used for stereotyped behaviours, such as a stereotyped manifestation of excessive discharge of cerebral neurones. Brown[29] in *An Epilepsy Needs Document* adopts the following definition:

'An epileptic seizure is a brief, usually unprovoked stereotyped disturbance of consciousness, behaviour, emotion, motor function or sensation...'

ISSUES IN CLASSIFICATION

In addition to overlap in terminology, both seizures and stereotyped behaviours exhibit a number of topographies, with seizures being classified according to external manifestations and (to a lesser degree) neurological site or origin (Appendix 1). As previously referred to, it is often the case in epilepsy that a diagnosis is required to be made which is based partly on descriptions of an external event which the specialist has never witnessed in that particular patient. This highlights the importance of paying attention to each specific body part included in these descriptions.

It is usually the case that seizure descriptions which incorporate shrieking, stiffening, falling to the ground, losing consciousness and jerking are assigned to the category of generalized events quite unambiguously. Similarly, observations of someone remaining conscious but jerking their arm and reporting a sensation of 'butterflies' in the stomach would initially be regarded as a partial seizure. With regard to stereotyped actions, they also have a considerable degree of variability, with individuals often becoming associated with their own distinct repertoire (Appendix 2). Dantzer[30] has suggested that there are numerous types of stereotyped behaviours yet only a small percentage are deemed to be 'stereotypies' and deciding where one finishes and the other starts appears to be a totally arbitrary decision. Therefore classification difficulties a bound.

A number of researchers[31,32] compiled inventories of the types of stereotypies displayed by adult learning disabled people and these have included rocking, complex movements of the hands and head banging. Some of these behaviours, particularly the elaborate hand and orofacial gestures, increasingly appear to resemble some of the motor manifestations of temporal lobe epilepsies.

Further difficulties are introduced by the fact that EEG changes are only apparent in 15–20% of simple partial events[34,34] therefore they may not be regarded as epileptic in origin following EEG investigations, yet may be genuinely epileptic. Also, ictal and interictal scalp EEGs do not change in individuals with frontal lobe epilepsy yet they may exhibit external motor behaviours with no loss of consciousness.

The International Classification of Seizures (*Epilepsia* 1981) describes partial events as follows:

Simple partial: consciousness is not impaired in these seizures and normal awareness is maintained. There is a great variety of these. There may be jerking of a limb, posturing, or numbness and tingling of a part of the body or sensations such as fear, a rising feeling in the stomach, deja vu (recall of past memories), or auditory, visual, gustatory (taste) and olfactory (smell) hallucinations.

Complex partial: in these there is alteration of normal alterness and awareness. Complex partial seizures may sometimes, but not always, start with a simple partial seizure then develop. During complex partial seizures there may be automatisms. Automatisms usually consist of repeated semi-purposeful motor actions such as chewing, lipsmacking, making brushing movements with the hands, fiddling with objects. Repeated speech, mumbling, walking about, incontinence and falling over may occur.

Witnessing the behaviour of an adult with a severe learning disability in a day centre may give rise to the observation of any number of the aforementioned behaviours in a relatively short space of time. As a result, there are often great

difficulties with such a client group in recording seizure occurrence. This is partly explained by the limitations in movement posed by secondary neurological impairment, individuals are thus able to move only a limited number of limbs and sometimes in a somewhat irregular fashion. This is frequently interpreted as involuntary movements or dyskinesias associated with movement disorders. However, in the absence of any clear diagnosis of movement disorder some of these external manifestations do prove difficult to classify and are likened to spontaneous epileptic phenomenon.

Perhaps the advantage which epileptic behaviours have over stereotyped movements is that, although wide-ranging, epileptic movements do have established criteria, e.g. a stiffening of limbs, jerking, appearing cyanosed, incontinent, which attach to some label, e.g. primary generalized, which in turn allows for an understanding of the function of the movement. What this means for the professional is that they can match descriptions to these pre-determined categories in order to make sense of the behaviour.

Stereotypies, on the other hand, appear to have an infinite number of possible manifestations which are extremely difficult to classify and although in some cases they are replicated across individuals, i.e. finger flicking, opinions on their functional perspective vary greatly.

Despite this fact, their overlap with external manifestations of epileptic activity deserves to be further explored for a number of reasons. As previously mentioned, frequency rates of both types of behaviour (epilepsy and stereotypies) are very high in this population but with no definite explanation. In addition, the neurological pathways for both——inferior parietal lobule, basal ganglia and brainstem—are frequently connected. Both behaviours have responded to pharmacological intervention although the mechanisms of action are often unclear, this will be explored in more detail further on. Finally both interfere, in some cases to a high degree, with an individual's quality of life and perhaps for that reason alone deserve exploration.

VIDEO AND EEG INVESTIGATIONS

Investigations of stereotyped and epileptic phenomenon in learning disabled adults have centred primarily around videotelemetry and other EEG investigations. This is ultimately because these procedures allow for the clinical detection and, to a lesser degree, explanation of such phenomenon.

In the early 1960s Stone[35] investigated EEGs of blind learning disabled children who engaged in such stereotypies. Tracings appeared to reveal a relationship between slowing of the wave patterns and typical 4–6 s waves during lengthy periods of 'blindism' (rocking and eye pressing) but not during more transient episodes. Stone interpreted this behaviour as the children's attempt to reduce arousal.

Also around this time Hutt and Hutt[36] paid particular attention to the clinical changes in the EEGs of autistic children during a wide range of motor behaviours. They implemented an extremely thorough methodology. They dictated commentaries during observations of behaviours which were then synchronized with EEG traces. The duration of activity in each of the four bands, delta, theta, alpha and beta was measured along with any low voltage irregular activity, but there was no evidence to indicate any specific EEG pattern during stereotypies.

Latterly however[37] they analysed the occurrence of stereotypy and low voltage activity in each specific situation. What did emerge was that as situations became more complex with more people in the room, increasing noise, numerous objects, both stereotypies and low voltage activity increased.

Other investigators[38], detected specific abnormal patterns in the EEG such as focal slowing, spiking or paroxysmal spike wave discharges again in autistic children however these were not linked to the actual displays of stereotypies. Donat and Wright[39] also implemented video-EEG techniques to assess symptoms which apparently imitated epileptic seizures in learning disabled children. Indeed in some cases the situation had been considered severe enough to commence the individual on antiepileptic medication. The main symptoms present are listed here:

(1) head movements; shaking, nodding; (2) eye movements; lateral/vertical nystagmus, deviations, (3) staring, (4) mouth movements; chewing, mouthing, twitching, tongue thrusting, (5) respiratory movements; hyperventilation, apnoea, ataxic/periodic breathing, (6) tonic posturing; opisthotonic, decorticate, decerebrate, asymmetrical tonic neck reflex, (7) coarse tremors, (8) sleep myoclonus, (9) other; tics, myoclonus, dystonia, excessive startles.

They concluded that in every case there was no corresponding change in the EEG during such movements, there was no evidence of epileptiform activity. Similar results were reported by Sassower and Duchowny[40] where particular in-

vestigations conducted during periods of self-stimulation revealed only movement artefact and myogenic potentials in young children with a learning disability.

More recently, Desai and Talwar[41] used similar techniques to explored the non-epileptic nature of particular behaviours in 27 children (age range, 0.1–19 years), nineteen of whom had a learning disability. They concluded that such phenomenon could be classified as any of the following; abnormal movements, conversion disorder, staring, sleep disorder, behavioural episodes and apnoea, with abnormal movements (58%) and staring (26%) being most commonly observed in those with a learning disability.

MANDY—CASE STUDY 1

Unfortunately EEG assessment may be even more complicated in people with multiple disabilities who have extremely limited motor movements. This is because generally diagnosis of epileptiform activity is assessed on two factors, (1) changes in the trace such as recognizable epileptiform activity and (2) external clinical manifestation of that activity. Where problems can arise is when movements are so subtle that they are difficult to detect and the individual has limited communication.

In this particular case clinical movements were confined to extremely subtle deviations of the eye. This was observed during a routine investigation where corresponding video investigations are not usually undertaken. The technician who was unable to observe the patient fully due to restricted physical movements of the patient and height of wheelchair concluded that:

'... alpha rhythm is absent from the trace. The dominant post central activity is in the theta waveband, varying in frequency and between 5 and 7 Hz. This is intermixed to a fairly high degree with delta activity. Against this background frequent episodes of sharp-and-slow, spike-and-slow or polyspike-and-slow wave activity occur bisynchronously over the hemispheres. These are without clinical accompaniment...'

Certainly the clinical opinion concentrated upon the neurophysiological evidence and concluded with '... most of the background changes are due to post-ictal effects... this indicates a poor seizure control.' Video illustrations may have been useful in this example for assisting clinical opinion.

What this case illustrates is that set criteria for different seizure types are useful and in fact correct in diagnostic assessment for the vast majority of people with epilepsy. Nevertheless, in some cases restricted movements and indeed communication abilities make it difficult for external manifestations to replicate those generally observed.

ANTIEPILEPTIC DRUGS

As with the general population, pharmacological interventions are usually adopted for epilepsy treatment. However a high proportion of the learning disabled population have seizures which are refractory to treatment, adding further complexity. Monotherapy alone is insufficient, necessitating the use of more than one antiepileptic with the increased potential for interactions. Thus the likelihood of side-effects will increase but in some cases unfortunately the individual is unable to communicate these to their carer. The result is that some people may be heavily sedated and unresponsive, but it is assumed to be a personality trait or even worse they may be displaying symptoms of toxicity which remain undetected for a considerable period of time.

DUAL PRESCRIBING–ANTIEPILEPTICS AND NEUROLEPTICS

To increase the complexity of the situation further, a high proportion of people with a learning disability are also in receipt of psychotropic, in particular neuroleptic, medication for behaviour problems or psychiatric illness. Rinck[42] estimates that between 20 and 50% of both community and institutional populations take regular doses of neuroleptics which compares with Fischbacher's[41] calculations of 20–45% of such people on antiepileptic drugs. In some cases antiepileptics have been used as dual purpose such as carbamazepine which is regularly prescribed for behaviour problems and epilepsy.

Although some antidepressants, anxiolytics and hypnotics may be contra-indicated in epilepsy, the focus here is upon neuroleptics. Unfortunately, despite the high prescribing rates, very little is known of the interaction, both pharmacokinetic and pharmacodynamic, of both categories of drugs, neuroleptics and antiepileptics, in learning disabled people. However the following information has been reported in the general population. Those drugs which indicate an apparent high risk for lowering seizure

threshold include loxapine and chlorpromazine with pimozide, sulpiride and fluphenazine being considered a low risk. Those which introduce a moderate level of risk are clozapine, haloperidol, risperidone, thioridazine and zuclopenthixol. An actual example of an individual in receipt of a moderate risk neuroleptic, thioridazine and an antiepileptic, carbamazepine, is presented in the following case study.

DAVID—CASE STUDY 2

David is a 22-year-old man with Down Syndrome and a severe learning disability who resided at home until leaving full-time education. He moved into a residential resource centre for 2 years, with frequent visits home, before finally being offered a place in a community staffed house where he has been for the last 2 years. Since his teenage years he has at times presented with behavioural problems. He has limited verbal communication, restricted to a few signs (often displayed out of context) and some vocalizations, but his verbal comprehension is good.

He has a history of complex partial epilepsy which was recently reported as increasing. Staff referred to at least fortnightly episodes lasting up to 9 hours where David displayed a wide range of stereotyped behaviours (which is a regular feature of David's behaviour). However these behaviours differed in that David's eyes would deviate to one side and he would thrust his fingers down his throat but most important of all, he was extremely compliant and would follow instructions without resistance.

Initially it was hypothesized that these displays were linked to some kind of status and he was referred for a routine EEG which was abandoned due to lack of compliance with the procedure, despite the usage of an electrode cap for ease of attachment. However, it had also been revealed that due to an increase in behavioural problems, David's neuroleptic medication had been doubled 8 weeks prior to the referral.

Following this, it was arranged for staff to call the EEG department if David displayed these behaviours and if possible to conduct an assessment immediately. This proved extremely successful and a much more compliant David received a routine EEG which concluded with: 'Apart from minor slowing of the background rhythms, there are NO epileptiform discharges in this EEG. This would make it unlikely that the 'absence' described is epileptic in nature.'

Staff therefore requested a further review of medication as it was considered that perhaps these behaviours (particularly orofacial) were the result of neuroleptic administration. This case again serves to highlight the difficulties involved in diagnosis on a population where communication can be extremely limited and dual prescribing can occur without standardized monitoring procedures.

CONCLUSION

The diagnosis and corresponding treatment of a wide range of medical conditions relies upon the recognition of common symptomatology. Occasionally difficulties arise when symptoms do not appear to be classic and this is frequently the case with learning disabled people and epilepsy. Confusion abounds with motor behaviours which mimic stereotyped movements, some associated with particular movement disorders. EEG techniques are primarily adopted to aleviate this difficulty. If diagnosis is clarified and pharmacological interventions are employed then the problems can be exacerbated by interactions with medications being taken for other behaviours, in particular neuroleptics prescribed for behaviour problems.

Detailed histories are vitally important for correct interventions, as are consistent monitoring techniques in a population who often cannot self report side-effects. What the physician may find difficult to establish is whether carers are reporting general behaviours (as there is a tendency to do) or those which specifically relate to possible side-effects of treatment.

OUTCOMES

The situation in these cases could be improved by establishing a sound battery of outcome measures for use in this population. This would include scales for subjective ratings of specific behaviors, concentrating on those which may genuinely be linked to pharmacological and physiological changes, such as drowsiness and loss of appetite. In addition, treatment monitoring could be more confidently undertaken as carers are sometimes unsure of what exactly to report.

Perhaps the design of specific cognitive testing material could be carried out, as in some cases (people with mild learning disabilities) it would be feasible to conduct and would allow a more

objective assessment to be made. The quantitative data recorded would be available for comparison. Increased usage of video material for recording events as they occur in a variety of settings is useful for assisting with differentiation of epileptic events from other behaviours.

Research priorities would also include further exploration and perhaps standardized coding of these motor phenomenon in this population using video material. This would involve recording the behaviour and retrospectively analysing it by assessing the environment, specified body parts involved in the behaviour, its duration, etc. in order to improve understanding.

As previously highlighted, combined video and EEG monitoring is a useful assessment procedure and could be implemented in learning disabled people with no previous history of epilepsy but displaying stereotypies/movement disorders. This would then provide comparative information on areas such as background activity, dominant rhythms and external behaviour.

Diagnosis may never be easy in this population but the implementation of specific investigation techniques as standard practice may at least provide valuable assistance to those working in this area.

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APPENDIX 1—CLASSIFICATION OF SEIZURES

Tonic–clonic

The most dramatic form is the generalised convulsion in which the person becomes rigid, falls to the ground and there is jerking of all four limbs. Breathing is laboured and may be interrupted, with cyanosis (blueness from lack of oxygen). There may be incontinence of urine. Not all of these features are always seen.

Clonic

There is jerking of all limbs without a prior period of stiffening.

Tonic

There is general stiffening of muscles without rhythmical jerking. The person may fall to the ground if standing, with consequent risk of injury.

Atonic

There is sudden loss of muscle tone, causing the person to become floppy and collapse to the ground.

Myoclonic

There is abrupt, sudden, brief jerking of one or more limbs. These often happen within a short time of waking up, either on their own or in association with other forms of generalized seizures.

Absences

There is a brief interruption of consciousness. Blank staring, fluttering of the eyelids and nodding of the head may occur.

Simple partial

Consciousness is not impaired in these seizures and normal awareness is maintained. There is a great variety of these, there may be jerking of a limb, posturing or numbness and tingling of a part of the body, or senstations such as fear, a rising feeling in the stomach, deja vu (recall of past memories) or auditory, visual, gustatory (taste) and olfactory (small) hallucinations.

Complex partial

In these there is alteration of normal alertness and awareness. Complex partial seizures may sometimes but not always, start with a simple partial seizure and then develop. During complex partial seizures there may be automatisms. Automatisms usually consist of repeated semi-purposeful motor actions such as chewing, lip-smacking, making brushing movements with the hands, fiiddling with objects. Repeated speech, mumbling, walking about, incontinence and falling over may occur.

Adapted from International classification of

seizures. Commission on classification and terminology; proposal for revised seizure classifications. *Epilepsia* 1981; **22**: 489–501.

APPENDIX 2

Range of stereotyped behaviours exhibited by four individuals with profound learning disabilities:

Stephen: body rocking, head jerking, arm waving, arm flapping, vocalizations.
Debbie: hand waving in front of face, finger flicking, body swaying, head jerking, shifting weight from foot to foot, posturing.
William: finger pointing, arm waving, excessive walking, vocalizing.
Jim: pacing, head jerking, pointing, vocalizing, hand flicking.